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Valvular Heart Disease

The Role of Cardiac Catheterization in Preoperative Evaluation

ARTHUR SELZER, M.D., San Francisco, and HERBERT W. BRADLEY, M.D., Yuba City

ACQUIRED VALVULAR HEART DISEASE is gradually entering the category of surgically correctible cardiac lesions. Recent strides in the development of surgical techniques for valvular repair suggest that the time may be near when a comprehensive cardiac valvuloplastic operation will be feasible. At present, however, the only operation that has been generally accepted and shown to give satisfactory results with low mortality rate is the correction of mitral stenosis by finger fracture or valvulotomy. The excellent results that may follow such an operation have been attested by clinical observations on large series of patients with mitral stenosis. However, it remained for the quantitative circulatory studies by means of cardiac catheterization to show conclusively the dramatic improvement in circulatory dynamics that may occur after operation, thereby satisfying skeptics that the clinical improvement is not due to prolonged bed rest and nonsurgical or extracardiac influences.

Thus, cardiac catheterization plays a major role in the assessment of operations on the heart. Its main value, however, lies in permitting the collection of hemodynamic data in a research laboratory in series of cases rather than its use as a diagnostic procedure in an individual case. This is largely due to the fact that in left sided valvular lesions cardiac catheteriza-

tion provides only inferential information and not direct diagnostic details which are so helpful in congenital cardiac defects affecting the right heart. The purpose of this discussion is to present briefly the contribution of cardiac catheterization to the diagnosis of left sided lesions, and to cite specific instances in which such a method may help to decide whether operation is indicated in an individual case.

The comprehensive catheterization study necessary for the assessment of valvular cardiac lesions can only be performed in a fully equipped research laboratory. It should be done in a condition as close to a basal state as possible, with preparations similar

Cardiac catheterization studies performed in research laboratories showed that advanced mitral stenosis is associated with a characteristic dynamic pattern which is reversible by mitral valvulotomy. In the process of the selection of patients for mitral valvulotomy, occasionally there are instances in which a decision cannot be reached on the basis of ordinary clinical methods of examination. In some such cases cardiac catheterization may be of decisive value by demonstrating, or by failing to demonstrate, the dynamic pattern of mitral stenosis. Cases in which this diagnostic procedure is most often helpful are those of mild mitral stenosis and those in which there are combined valvular defects.

From the Medical Service, Veterans Administration Hospital, and the Department of Medicine, Stanford University School of Medicine. Supported by a grant from Monterey County Heart Association.

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to those for a standard basal metabolism test. The essential equipment includes a reliable recording system capable of reproducing graphically undistorted pressure tracings. Oxygen consumption should be measured by the analysis of expired air and the total ventilation and respiratory quotient included in the measurements. A simple and readily reproducible exercise test lasting seven to ten minutes should be available to be performed by the patient in recumbent position. Pressure tracing should be recorded from the "wedge" position* in the pulmonary arterial branch simultaneously with an electrocardiographic tracing for the purpose of timing. Pulmonary arterial pressure should be observed before and during exercise. Right ventricular and right atrial curves should be recorded with special care in assessing the "zero" reference point in relation to the thorax. Determination of cardiac output by the Fick principle should be made before exercise and during the last three minutes of the exercise period with samples of mixed venous blood withdrawn from the main pulmonary artery.

It is generally agreed that in pure mitral stenosis cardiac catheterization is not necessary as a routine preoperative procedure. However, there are situations in which the degree of mitral stenosis has to be assessed by more accurate procedures than the routine physical examination and electrocardiographic and roentgenographic evaluation. In such instances cardiac catheterization frequently becomes the method of choice.

The average hemodynamic findings in a typical case of mitral stenosis and the change which takes place after successful valvulotomy are presented in the following case summary.

CASE 1. A 37-year-old man had pronounced limitation of activities due to dyspnea and tiredness. He could only engage in semisedentary work. Upon physical examination findings typical of mitral stenosis were noted—a loud diastolic and presystolic rumbling murmur in the apical region of the heart, with a loud first sound in this area, a prominent mitral opening snap and an accentuated and reduplicated second sound at the left upper sternal border. An electrocardiogram revealed a vertical rotation of the heart with prominent R-waves and inverted Twaves in precordial leads V1 and V2, suggestive of an enlarged right ventricle, and tall and bifid P-waves. A roentgenogram of the thorax showed no generalized cardiac enlargement but there was evidence of dilation of the left atrium and of the pulmonary artery. The hemodynamic data are presented in Chart 1, where it can be seen that moderately severe pulmonary hypertension, low cardiac output (cardiac index) and increased arteriovenous difference were present. During exercise there was a decrease, instead of an increase, in the cardiac index. Such

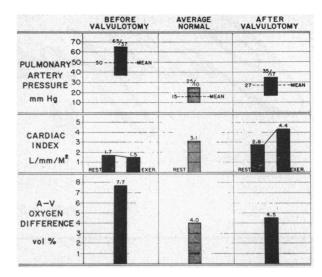


Chart 1.—Graphic presentation of the essential findings on cardiac catheterization before and after mitral valve operation in Case 1. The central column represents average normal values.

findings, with the addition of an elevated pressure reading in the pulmonary wedge position (which was not obtained in this case) can be considered as characteristic for advanced mitral stenosis. The patient underwent mitral commissurotomy and the tolerance for exercise was so much increased after the operation that he could do strenuous work ten hours a day without discomfort.

Upon examination a year after operation the heart sounds and murmurs were essentially unchanged from those heard preoperatively. However, the electrocardiographic evidence of right ventricular hypertrophy was no longer present and in a roent-genogram of the chest the left atrial and pulmonary arterial segments appeared more nearly normal. Hemodynamic data (Chart 1) showed only slightly elevated pulmonary arterial pressure, a cardiac output (index) well within normal limits, and a normal increase in circulation on exercise.

It appears from the foregoing presentation that an eminently successful operation reverted the circulatory changes of advanced mitral stenosis into those of mild stenosis. The persistence of auscultative symptoms and the mild elevation of the pulmonary arterial pressure leaves no doubt that a narrowing of the mitral orifice was still present. However, barring restenosis of the mitral orifice, the degree of circulatory derangement could well be consistent with a normal or almost normal life expectancy and a reasonably active life. This is worthy of emphasis in view of some diagnostic difficulties which may arise in milder cases of mitral stenosis, as is exemplified in the following case.

CASE 2. The patient, a 30-year-old woman, said she had had limitation of activities consisting of a feeling of exhaustion associated with some shortness of breath both at rest and during exercise for a

^{*}The catheter wedged firmly into a smaller branch of the pulmonary artery records not pulmonary arterial pressure but a pressure curve reflecting the dynamic events in the left atrium.

TABLE 1.—Cardiac catheterization findings in Case 2

Pulmonary wedge pressure	9 mm. mercury
Pulmonary artery at rest	
	(mean 16)
Pulmonary artery during exercise	
	(mean 17)
Right ventricle	20/2 mm. mercury
_	(mean 10)
Cardiac output (index) at rest	3.7 liters per min.
	$dex 2.5 lit/min/m^2$
Cardiac output (index) during exercise	4.5 liters per min.
	dex 3 1 lit/min/m²)

TABLE 2.—Cardiac catheterization findings in Case 3

Pulmonary wedge pressure	30 mm.mercury (mean 30)
Pulmonary artery at rest	• • • • • • •
Pulmonary artery during exercise	69/36 mm. mercury (mean 55)
Right ventricle	55/8 mm. mercury (mean 29)
Cardiac output at rest	2.9 liters per min. (1.4 lit/min/m²)
Cardiac output during exercise	4.3 liters per min. (2.2 lit/min/m²)

period of over three years. Upon auscultation the classical findings of mitral stenosis were notednamely, a loud diastolic rumbling apical murmur with a presystolic accentuation, loud first sound, an opening snap and an accentuated second pulmonary sound. A roentgenogram of the chest showed mild enlargement of the shadow of the left atrium. An electrocardiogram showed a normal record with a bifid P-wave. Cardiac catheterization was performed with results shown in Table 1. Intracardiac, pulmonary arterial and wedge pressures were entirely normal and the cardiac index was within normal limits and increased with exercise. The clinician evaluating this case had some doubt in accepting at face value the patient's story of limitation of activities. The data obtained on cardiac catheterization proved of great value, for they permitted the conclusion that authentic cardiac symptoms were absent or unimportant and that tiredness and "shortness of breath" were in reality manifestations of neurocirculatory asthenia. Obviously, surgical therapy of mitral stenosis was not indicated even though the classical physical findings of pure mitral stenosis were present.

Mitral insufficiency presents an important problem in the preoperative evaluation of patients with rheumatic heart disease. Severe mitral insufficiency constitutes a contraindication to mitral valvulotomy. The diagnosis of mitral valve disease with predominant mitral insufficiency may be easy in its typical form, when a loud apical systolic murmur is heard and is conducted to the left scapula, and when evidence of left ventricular enlargement is found upon physical examination and by electrocardiogram and roentgenogram. However, in many instances the unknown extent of mitral insufficiency in a case of mitral stenosis severe enough to warrant surgical consideration may present great difficulty. In such cases supplementary information is sought by a fluoroscopic or kymographic study of the motion of the left atrium during systole, by cardiac catheterization or by angiocardiography. Cardiac catheterization may on occasion supply definitive information, which is exemplified by the following case.

CASE 3. A 43-year-old man with rheumatic heart disease had moderate to severe limitation of activities due to dyspnea. Some months before the present hospital admission, there was a bout of cardiac failure which was promptly controlled by the use of digitalis and a brief course of mercurial diuretics. Upon auscultation a long and loud rumbling apical diastolic murmur, a mitral opening snap and a reduplicated second pulmonic sound were noted. In addition there was a moderately loud systolic murmur which was heard at the apex and along the sternal border up to the base of the heart. At the lower left sternal border a soft blowing early diastolic murmur was also heard. The blood pressure was normal. An electrocardiographic tracing was suggestive of hypertrophy of both ventricles. A roentgenogram of the chest showed an enlarged left atrium, a moderately large pulmonary artery segment and an enlargement of both cardiac ventricles.

Cardiac catheterization was performed with the results shown in Table 2. Elevated wedge pressure and moderately elevated pulmonary artery pressure with considerable increase on exercise were indicative of mitral stenosis of considerable severity. However, a pressure tracing from the pulmonary wedge position (Figure 1) showed a prominent systolic wave which was thought to be caused by significant mitral regurgitation. Furthermore, it was noted that the cardiac output was very low but increased in a normal manner with exercise. Such a response is seldom seen in "tight" mitral stenosis where mechanical obstruction limits the flow through the mitral orifice (see Chart 1). Thus the data obtained by cardiac catheterization suggested that in this case mitral insufficiency was not only present but that its effect predominated in the dynamic pattern of the circulatory derangement. On the basis of these findings it was felt that the patient probably would not benefit from operation on the mitral valve, and could easily be made worse.

CASE 4. A somewhat similar clinical problem was present in the case of a 39-year-old man whose activities were severely curtailed by dyspnea and weakness and who was gradually becoming worse.

An apical diastolic rumbling murmur was heard, with an accentuated first sound. The second pulmonary sound was very loud. A moderately loud systolic and a faint early diastolic murmur were heard at the lower left sternal border with the systolic murmur conducted to the left axilla on one hand, and to the base of the heart and the great vessels on the other hand. A totally irregular pulse was noted. The pulse pressure was normal. An electrocardiogram revealed atrial fibrillation and a "balanced" pattern

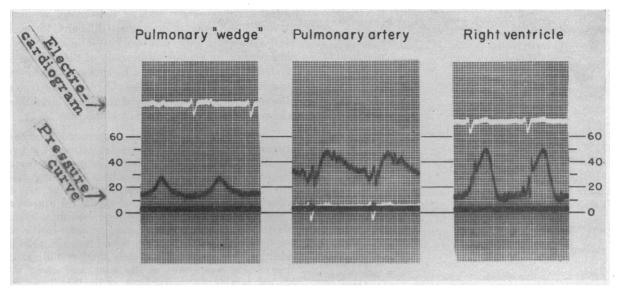


Figure 1.—Reproduction of the pressure tracings from the pulmonary "wedge" position, the main pulmonary artery and the right ventricle in Case 3. The high systolic wave in the pulmonary wedge tracing is characteristic of mitral insufficiency.

of the ventricular complexes suggesting hypertrophy of both ventricles. A roentgenogram showed enlargement of all the cardiac chambers.

Cardiac catheterization (Table 3) showed moderate to severe elevation of pressure in the pulmonary wedge position, in the pulmonary artery and the right ventricle. A steep further increase in pressure occurred upon exercise. The cardiac output was very low and showed an insignificant increase on exercise. It was thought in this case that the dynamic pattern of mitral stenosis predominated to such an extent that mitral valvulotomy might reasonably be expected to relieve some of the disability, and in view of the progression of symptoms the surgical risk appeared justified. Mitral valvulotomy was performed and tight mitral stenosis and a mild regurgitant jet were observed.

The illustrative case summaries presented cite actual instances in which cardiac catheterization played a major role in deciding whether or not cardiac operation was advisable in a given patient. They exemplify the main problems in which help can be expected from this procedure. It is obvious that cardiac catheterization is not necessary in the average case of mitral stenosis in which the lesion has led to the appearance of incapacitating symptoms. The effect of mitral stenosis upon cardiodynamics is well enough known so that alteration of pressures and flows can roughly be predicted from the results of conventional methods of examination. Since cardiac catheterization does not directly prove or disprove the presence of mitral stenosis (the evidence is not conclusive as it is in pulmonary stenosis), one has to rely on the pattern of findings usually associated with significant degree of mitral valve narrowing. This "hemodynamic pattern" of mitral stenosis is

TABLE 3.—Cardiac catheterization findings in Case 4

Pulmonary wedge pressure	25 mm. mercury (mean 25)
Pulmonary artery at rest	
Pulmonary artery with exercise	
Right ventricle	
Cardiac output at rest	
Cardiac output during exercise	

TABLE 4.—Hemodynamic pattern of mitral stenosis

	mm. Mer- cury	Liters min./ m.²	— Nor mm. Mer- cury	mal — Liters min./ m.²
Pulmonary wedge pressure Systolic wave			5-10	
Pulmonary artery pressure: Systolic Mean	< 50		20 12	
Cardiac index		1.5-2	12	3.1
Effect of exercise: (a) artery pressure; (b) Static			in pub	monary

summarized in Table 4. The pattern is of course not specific for mitral stenosis; each of the components occurs in other forms of cardiac disease.

Pulmonary wedge pressure constitutes the most important diagnostic finding associated with tight mitral stenosis. It has been shown that pressure reading and the shape of the curves obtained from the pulmonary wedge position reflect closely the dynamic events in the pulmonary veins and in the left atrium. Thus, pulmonary wedge pressure is elevated in mitral stenosis, in left ventricular failure and in left sided constrictive pericarditis. Since the

latter two conditions can usually be eliminated diagnostically on clinical grounds, elevation of pulmonary wedge pressure constitutes not only an important confirmatory finding of mitral stenosis, but a rough index of its severity. Conversely, the absence of elevation of pulmonary wedge pressure makes the diagnosis of significant mitral stenosis untenable. The finding of pulmonary hypertension with a normal pulmonary wedge pressure proves that pulmonary resistance is elevated at the level of the pulmonary arterioles, and in such circumstances clinical signs of mitral stenosis cannot be considered to be of surgical importance. The effect of mitral regurgitation upon the pulmonary wedge pressure is the appearance of a prominent systolic wave of more than 5 mm. of mercury. In severe mitral insufficiency the wedge pressure curve may exhibit a pulse pressure of over 20 mm. of mercury. The diastolic part of the curve shows the pressure to be normal unless significant mitral stenosis is also present or left ventricular failure ensues. While the presence of a significant wave of mitral insufficiency is of great diagnostic importance, its absence does not rule out mitral regurgitation.

Pulmonary hypertension occurs as a rule in significant mitral stenosis but is of no diagnostic importance because of its common occurrence in other cardiac conditions. The degree of pulmonary hypertension, however, permits distinction between early cases, in which moderate elevation of pulmonary arterial pressure is found, and advanced cases in which severe pulmonary hypertension indicates secondary changes in the pulmonary arterioles. The first form may be completely reversible by mitral valvulotomy, while the second form is only partially reversible. In mitral stenosis pulmonary arterial pressure almost always rises during exercise.

Low cardiac output with the patient at rest is found in the majority of cases of mitral stenosis. Often this phenomenon occurs early in the course of the disease, when pressures are only mildly elevated. It is, however, found also in most forms of cardiac failure and is therefore a non-specific finding. More important than the finding of a low resting output is the response of output to exercise. In cardiac failure the cardiac output increases on exercise (although not as much as in health) with the exception of some cases of very severe cardiac insufficiency. As a rule, static cardiac output means that a mechanically limiting factor exists within the heart and therefore points to severe stenosis of one of the four cardiac orifices. The static cardiac output in severe mitral stenosis is reversible by operation on the mitral valve (Chart 1).

The data obtained from the previously mentioned measurements during cardiac catheterization permit an estimation of the size of the mitral orifice by available formulae. However, the accuracy of these formulae has not been generally accepted and the numerical expression of the size of the mitral orifice as thus computed may be irrelevant once the diagnosis of tight mitral stenosis is established and the indication for operation ascertained.

Thus, the comprehensive pattern of the effect of mitral stenosis upon cardiodynamics may be utilized in doubtful cases in presurgical evaluation. As exemplified by the three case summaries, aid obtained from cardiac catheterization is greatest in two categories of cases. The first category is mild mitral stenosis, where the extent of disability and its connection with the valve defect cannot be determined with certainty by ordinary clinical means. In such cases normal or almost normal cardiac dynamics make it most unlikely that the patient suffers from the result of mitral valve obstruction, and therefore unlikely that operation on the valve would be of benefit. Conversely, the dynamic pattern of significant mitral stenosis exists occasionally in cases in which cardiac strain and enlargement are not yet recognizable by electrocardiographic and roentgenographic changes. In such instances mitral stenosis may cause incapacitating symptoms which might be relieved by operation even though clinical findings as regards the heart are within normal limits and lead to erroneous conclusion that symptoms are extracardiac.

In combined valvular lesions it is important to determine whether mitral stenosis is the predominant lesion and is primarily responsible for the incapacitating symptoms, for in such cases mitral valvulotomy can be of benefit. In patients with combined valvular defects various findings of the catheterization study may be used in differential diagnosis. The most important feature of the dynamic pattern of mitral stenosis in such cases is the low and static cardiac output, which, in the absence of severe aortic stenosis, strongly suggests tight mitral stenosis, for usually in mitral or aortic insufficiency there is some increase of blood flow on exercise. Elevated pulmonary arterial pressure may be present in mitral insufficiency or in aortic valve defects combined with left ventricular failure. However, severe pulmonary hypertension is strongly indicative that mitral stenosis is predominant. Finally, pulmonary wedge pressure may reveal the presence of mitral insufficiency.

Cardiac catheterization is a complex diagnostic procedure that should be performed primarily in a research laboratory. Routine use of the procedure as an aid to preoperative diagnosis in valvular heart disease is neither indicated nor desirable. It does, however, provide a way to get information, in cases in which diagnosis is in doubt, that cannot be obtained by any other means, thus permitting the preoperative assessment of some such cases.

450 Sutter Street.